Case # 10/549707 877/16/07-AD

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=> s monocyte and multipotent cell L1 46 MONOCYTE AND MULTIPOTENT CELL

=> s l1 and cd14

L2 2 L1 AND CD14

=> s 12 and cd34

L3 1 L2 AND CD34

=> s 13 and cd45

L4 0 L3 AND CD45

=> s 11 and cd45

L5 1 L1 AND CD45

=> s l1 and collagen type I

L6 0 L1 AND COLLAGEN TYPE I

=> s 12

L7 2 L2

=> s 12 and collagen

L8 0 L2 AND COLLAGEN

=> s monocyte and collagen j

L9 0 MONOCYTE AND COLLAGEN J

=> s 11 and fibronectin

L10 0 L1 AND FIBRONECTIN

=> s l1 and osteoblast

L11 1 L1 AND OSTEOBLAST

=> disp 111 ibib abs 1-1

L11 ANSWER 1 OF 1 SCISEARCH COPYRIGHT (c) 2007 The Thomson Corporation on STN

ACCESSION NUMBER: 2000:394976 SCISEARCH

THE GENUINE ARTICLE: 316VG

TITLE:

On the track of a human circulating mesenchymal stem cell

of neural crest origin

AUTHOR:
CORPORATE SOURCE:

Labat M L (Reprint); Milhaud G; Pouchelet M; Boireau P Ecole Natl Vet, INRA, AFSSA, INRA, UMR 956, 7 Ave Gen

Gaulle, F-94704 Maisons Alfort, France (Reprint); Ecole Natl Vet, INRA, AFSSA, INRA, UMR 956, F-94704 Maisons Alfort, France; CHU St Antoine, Dept Biophys, F-75012

Page 1

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Paris, France; INSERM, Serv Audiovisuel, F-78116 Le

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ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

The neural markers present in the normal circulating monocytoid cells able, in pathological situations, to transdifferentiate into different mesenchymal-type cells, confirm the hypothesis previously raised that AB these cells derive from the neural crest. In culture, the normal cells display a great plasticity very reminiscent of microglial cells in culture. Almost a quiescent cell in normal individuals, this monocytoid cell shows its division potentialities in pathological situations of fibrosis and cancer (chondrosarcoma) where it is found to spontaneously proliferate. While the normal neofibroblasts are rapidly recognized and destroyed by fibrophagic T-lymphocytes, the pathological cells escape this control and, as a result, they accumulate in vitro giving rise to a tissue sometimes organized as nodules. Although basically the sometimes organized as nodules. Although basically the transdifferentiation process is similar in all the pathological situations of fibrosis and cancer studied so far, the end-result phenotype evokes the pathology the patient is suffering from. It evokes ***osteoblasts*** in a case of osteomyelosclerosis, chondroidocytes in a case of chondrosarcoma, myelofibroblasts in a case of fibrosis of lung and kidney in a patient under ciclosporine treatment. Hence, this circulating monocytoid cell is a ***multipotent*** ***cell*** with great division potentiality. These are characteristics of stem/preprogenitor cells. Since this circulating monocytoid cell also bears the neural markers we called it a monocytoid ectomesenchymal stem/preprogenitor cell. Therefore, the existence of an ectomesenchymal system is discussed here. Therefore, the existence of an ectomesenchymal system is discussed here. The circulating monocytoid ectomesenchymal stem/proprogenitor cell might be involved in the normal cicatrisation process while the fibrophagic T lymphocytes might be involved in its termination. Impairment of this controlled mechanism might result in the development of fibrosis and/or cancer such as chondrosarcoma in vivo. Interestingly, at least in vitro, proliferation is restricted to the monocytoid cell before transdifferentiation takes place. In this model, fibrosis and cancer might share some common steps going from the proliferation of the monocytoid cells to their transdifferentiation into mesenchymal-type cells and the accumulation of these transdifferentiated cells in the tissues. Then, cancer might be distinguished from fibrosis by the additional acquisition of the ability to proliferate by the transdifferentiated acquisition of the ability to proliferate by the transdifferentiated cells. The monocytoid ectomesenchymal stem/preprogenitor cell might also hi: involved in brain neurodegenerative diseases characterized by an accumulation of microglia. The circulating monocytoid ectomesenchymal stem/preprogenitor cell appears as a target for gene therapy in pathological situations of fibrosis and/or cancer where it proliferates out of control. If the normal cell can be expanded and if its transdifferentiation can be directed, the circulating monocytoid ectomenschymal stem/ preprogenitor cell may become a useful tool for cellular therapy, in case of failure in wound healing and tissue cellular therapy, in case of failure in wound healing and tissue regeneration. (C) 2000 Editions scientifiques ct medicales Elsevier SAS.

=> FIL .DUTTA COST IN U.S. DOLLARS

ENTRY SESSION 21.36 21.57

FULL ESTIMATED COST

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=> s 11 and adipocyte 0 L1 AND ADIPOCYTE

=> s monocyte and multipotent cell L13 46 MONOCYTE AND MULTIPOTENT CELL

=> s 113 and adipocyte 0 L13 AND ADIPOCYTE

=> s 113 and myoblast **L15** 1 L13 AND MYOBLAST

=> s 113 and chondrocyte L16 0 L13 AND CHONDROCYTE

=> s 113 and myocardia L17 O L13 AND MYOCARDIA

=> disp 115 ibib abs 1-1

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2006:394620 SCISEARCH ACCESSION NUMBER:

THE GENUINE ARTICLE: 033PH

Multipotent ***cells*** of monocytic origin TITLE:

improve damaged heart function

Dresske B (Reprint); El Mokhtari N E; Ungefroren H; Ruhnke **AUTHOR:**

M; Plate V; Janssen D; Siebert R; Reinecke A; Simon R;

Fandrich F

Univ Schleswig Holstein, Dept Gen & Thorac Surg, Campus CORPORATE SOURCE:

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ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

Recently, we generated cells with multipotent properties from blood ***monocytes*** that in vitro differentiate into various somatic cel that in vitro differentiate into various somatic cell

types. This experimental study investigated whether these programmable cells of monocytic origin (PCMO) succeed to restore left ventricular function after myocardial infarction (MI). PCMO were generated from ***monocytes*** by exposition to RPMI medium containing M-CSF and IL-3 for 6 days. MI was induced in female Lewis rats ligating the left coronary artery. PCMO of male Lewis roughly (i. my) or intraverse of the coronary artery. intramyocardially (i.my.) or intravenously (i.v.) 24 h or 6 days post-infarction. Hemodynamic assessment after 60 days demonstrated significant improvement of left ventricular function following i.my. transplantation of PCMO as well as early (24 h post-infarction) i.v. application while nonmodulated ***monocytes*** failed to restore failed to restore heart function. The Y-chromosome-specific SRY gene of male donor PCMO was detected exclusively in infarcted hearts of animals, which demonstrated improved cardiac function. Subdivision of infarcted hearts by microdissection localized the SRY gene-containing department to the left ventricle adjacent to the infarcted area whereas the right ventricle remained negative. Successful generation of PCMO in access numbers allows their autologous use as a new additive treatment for early restoration of cardiac function after MI.